

glomerular injury [5]. It could be supposed that tubulointerstitial inflammation yields cytokines, growth factors, vasoactive molecules, and free oxygen radicals that contribute to the development of arteriolar and glomerular lesions. Some authors go further and extend this hypothesis, assuming that subtle interstitial injury is not so much the manifestation of hypertensive kidney disease, but rather the underlying reason for hypertension [6]. Further studies are necessary to clarify this issue.

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Reply from the Authors

Our paper [1] focused primarily on the relationship of arteriolar disease and tubulointerstitial inflammation in renal progression. With renal mass reduction there is an increase in systemic blood pressure with glomerular hyperfiltration, glomerular hypertension, and proteinuria. Tubulointerstitial inflammation results and appears to mediate afferent arteriolar disease, which further impairs autoregulation, resulting in further increases in glomerular pressure and acceleration of glomerulosclerosis. Arteriopathy in other conditions is also associated with glomerular hypertension, reduced plasma flow, ischemia, and fibrosis.

Drs. Tylicki and Rutkowski ask about the relationship of arteriopathy and tubulointerstitial inflammation in essential hypertension. This is an area of special interest to us because we have proposed that these changes are responsible for the development of salt-sensitive hypertension [2, 3]. As we have proposed, renal injury is initiated by vasoconstriction and glomerular hypertension induced by sympathetic nervous system overactivity, endothelial dysfunction, hyperuricemia, or activation of the renin-angiotensin system; this leads to tubulointerstitial inflammation, intrarenal oxidant and angiotensin II generation, and afferent arteriolar disease [4]. The arteriopathy results in impaired autoregulation and wors-

ens glomerular hypertension, and also decreases renal plasma flow, causing ischemia, thus perpetuating tubulointerstitial inflammation [2]. The observation by Drs. Tylicki and Rutkowski of urinary excretion of tubular markers in untreated hypertension [4] is consistent with our pathway. Thus, salt-sensitive hypertension may be considered a type of subtle acquired renal disease, which targeted therapies could possibly reverse and/or cure.

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GAPDH as housekeeping gene at renal level

To the Editor: In mRNA quantitative studies, choosing a reliable internal control is mandatory for monitoring intersample variations and comparing target RNA levels if assays are performed in different experimental sessions and come from different centers.

In microdissected kidney biopsies Schmid *et al* [1] studied the validity of three housekeeping genes commonly used in quantitative mRNA analysis: glyceraldehyde-3-phosphate dehydrogenase (GAPDH), 18S rRNA, and cyclophilin A. The authors demonstrated that in the tubulointerstitium of 12 out of 165 microdissected specimens, GAPDH expression was significantly lower in comparison to 18S rRNA or cyclophilin A, thus warning investigators on the use of GAPDH in this type of setting. Nevertheless, our experience in renal biopsies and in *in vitro* studies suggests the possibility of using GAPDH as internal standard.

We had the opportunity to study by real-time reverse transcription-polymerase chain reaction (RT/PCR) the